SYMPOSIUM: OPTIMIZING ENERGY NUTRITION FOR REPRODUCING DAIRY COWS

Influence of Supplemental Fats on Reproductive Tissues and Performance of Lactating Cows[1]

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ABSTRACT

Fat supplementation (about 3% of dietary dry matter) has often positively influenced the reproductive status of the dairy cow, including increased size of the ovulatory follicle, increased numbers of ovarian follicles, increased plasma concentration of progesterone, reduced secretion of prostaglandin metabolite, increased lifespan of the corpus luteum, and improved fertility. Supplemental fat may allay partially negative energy status during the early postpartum period, yet often the positive reproductive influence of supplemental fat has been independent of the energy status of the cow. The fatty acid profile of supplemental fats is influential to their impact. Linoleic acid and eicosapentaenoic acid (found in fish meal) are proven inhibitors of cyclooxygenase in endometrial tissue of dairy cows. As a result, endometrial secretion of PGF_{α} can be suppressed, thus potentially preventing early embryonic death. This process may be aided by the effect fat has in suppressing estradiol-17β secretion, thus reducing uterine $PGF_{2\alpha}$ secretion and decreasing the sensitivity of the corpus luteum to $PGF_{2\alpha}$. Targeting of dietary fatty acids toward ovarian and uterine function may enhance efficiency of reproductive management and fertility.
(**Key words**: fat, progesterone, prostaglandin, reproduction)

Abbreviation key: **BCS** = body condition score, **Ca-LCFA** = calcium soaps of long-chain fatty acids, **CL** = corpus luteum, **ES** = energy status, **HDL** = high density lipoproteins, **PGFM** = 13, 14-dihydro-15-keto $PGF_{2\alpha}$ metabolite, **PP** = postpartum.

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INTRODUCTION

Often the amazingly complex lactating dairy cow is studied compartmentally rather than holistically. The physiology of digestion, the endocrinology of ovulation and conception, or the biochemistry of milk synthesis can be studied as sequestered entities that function separately rather than as interdependent components of an organism. A disjointed understanding of the cow can occur if areas are studied in and of themselves; that is, specialization can lead to fragmentation of knowledge. The word "university" is derived from word roots suggesting unity from diversity. Frequently, communication among the ruminant nutritionist, the reproductive physiologist, and the veterinarian is limited. As a result, problems may not be addressed adequately, and progress in domestic livestock production may be hampered. The integration of the diverse disciplines of nutrition and reproduction often can lead to significant advances more quickly than study involving either discipline alone.

The influence of dietary fat supplements on reproductive performance is not well understood. Much of the published data comes from studies having nutritional rather than reproductive objectives. The studies often have been designed primarily to examine supplemental fat effects on DMI, digestibility, and milk production and composition, not on return to estrus, follicle growth, and pregnancy rates. As a result, many uncontrolled management factors have influenced the dependent variables of reproduction in these studies. Barton and Carroll (5) outlined the proper controls and criteria to include in the design of nutrition and reproduction experiments. Because of the large number of cows needed per treatment to ensure a reasonable chance of detecting small differences between treatments in variables such as conception and pregnancy rates, more of these studies need to be carried out on large commercial dairy farms. However, in such situations, the experimenter usually has less control of management practices

than on university farms. Despite these shortcomings, the existing data at this point need to be examined to determine the research priorities for the future. A commendable review of supplemental fat effects on reproductive performance of dairy cattle was published in 1991 by Grummer and Carroll (41). New information has been published since then, both on reproductive performance and possible mechanisms of action.

FAT SOURCES AND THEIR DELIVERY TO THE SMALL INTESTINE

For lactating dairy cows, most diets without supplemental fat contain approximately 2% long-chain fatty acids that are predominantly polyunsaturated. The major fatty acid in most seed lipids (e.g., corn and soybean meal) is linoleic acid ($C_{18:2}$); linolenic acid ($C_{18:3}$) predominates in most forage lipid (76). Linoleic and linolenic fatty acids are classified as essential fatty acids and must be supplied in the diet because the double bonds between the Δ-9-carbon and the terminal methyl group of a fatty acid cannot be inserted by mammalian biological systems.

Supplemental fats often are included in the diet of the early postpartum (**PP**) dairy cow in order to increase the energy density of the diet to attempt to meet the energetic demands of lactation. This practice is preferred to that of increasing the starch content of the diet, which can result in negative effects on digestion, milk composition, and health.

Many different types of supplemental fat have been fed to lactating cows under experimental conditions. Some of these include blends of animal-vegetable fat (74), tallow (93), yellow grease (28), oil in fish meal (17), whole oilseeds [cottonseeds (49), soybeans (70), rapeseeds (72), canola seeds (59), peanut hearts (57), safflower seeds (97), sunflower seeds (97)], flaked fat (31), prilled fat (56), hydrogenated fat (30, 54), calcium soaps of fat (35), medium-chain triglycerides (42), and free fatty acids (55). Thus, the availability of fat sources is quite diverse.

The fatty acid makeup of these fat sources varies widely. Coppock and Wilks (25) specified the fatty acid profile of many of the commonly used fats. As stated previously, many of the naturally occurring, unprocessed plant oils contain a large proportion of long-chain, polyunsaturated fatty acids, such as linoleic acid. The rendered fats, such as tallow and yellow grease, contain a large proportion of the monounsaturated fatty acid, oleic acid ($C_{18:1}$). Tallow can vary greatly in the ratio of saturated to unsaturated fatty acids and in the proportion of linoleic acid (range of 2 to 9%). Unfortunately, most published studies in which tallow was fed did not report the fatty acid profile of that tallow source. Granular fats, such as calcium soaps of palm oil fatty acids and prilled fats, contain mainly the saturated fats, palmitic and stearic acids.

The fate of these saturated fats in the rumen is well documented. Approximately 60 to 90% of unsaturated fatty acids in crushed rapeseed were biohydrogenated in the rumen before reaching the small intestine for absorption (72). In vitro biohydrogenation of continuously infused linoleic acid averaged 77% (34). Klusmeyer and Clark (60) calculated the biohydrogenation of unsaturated C_{18} fatty acids to be approximately 70% using ruminally and duodenally cannulated lactating dairy cows. It has been reported that ruminal microorganisms may incorporate linoleic acid and other fatty acids into their cellular lipids (7). Therefore, microbial biohydrogenation is not 100% efficient. Approximately 25% of the consumed unsaturated fatty acids may be available for absorption at the small intestine of the lactating dairy cow for delivery to tissues for metabolism. Essential fatty acids also may be made available for absorption from feeding ruminally inert fats (e.g., Megalac®, containing 8.5% linoleic acid; Church and Dwight, Piscataway, NJ).

Using certain assumptions of intake and biohydrogenation, the amounts of linoleic acid delivered to the small intestine by supplemental fat sources ranges from approximately 2 to 120 g/d per cow (Table 1). If linoleic acid plays a unique role in reproductive function for the lactating dairy cow, these small amounts delivered to the small intestine indicate its potency.

TABLE 1. Estimated delivery of linoleic acid to the small intestine.

Fat source	Fat source fed	Linoleic acid fed[1]	Linoleic acid appearing in small intestine[2]
	(kg/d)	(g/d)	
Whole cottonseeds	2.8	300	30 to 120
Whole soybeans	2.8	300	30 to 120
Yellow grease	0.45	77	8 to 31
Tallow	0.45	23	2 to 9
Megalac®[3]	0.45	38	25 to 34[4]

[1]Based on typical concentration of linoleic acid in fat source.

[2]Assumes microbial biohydrogenation in the rumen of linoleic acid in whole seeds and rendered fats to be 60 to 90% (72).

[3]Manufactured by Church & Dwight (Princeton, NJ).

[4]Assumes microbial biohydrogenation in the rumen of linoleic acid to be 33% (60) and 10% (98).

TABLE 2. Effects of supplemental fat on reproductive performance of lactating dairy cows.

Reference	Treatment[1]	Treatment period	Cows per treatment	DMI	Production of milk or FCM	Conception rate at first AI		Overall pregnancy rate	Overall conception rate	Days open	AI per Conception
		(DIM)	(no.)	(kg/d)		(%)					
(81)	0% Heated SB	1 to 105	27	22.7	33.5					115	2.1
	12.9% Heated SB		20	22.4	34.7					109	1.8
(84)	No oilseeds	28–112	75	20.8	31.4					126	2.15
	Oilseeds		78	20.4	30.9					136	2.38
(48)	0% Fat	1–112	18	18.2	25.9	35		83	88	107	1.87
	15% WCS		19	17.9	26.5	50		79	83	96	1.87
	WCS + 540 g/d of IF		19	18.0	26.1	44		79	83	123	2.47
(85)	0 g/d of IF	<30 to ≤ 140	54		34.5	43		72			2.3
	500 g/d of IF		54		36.2**	60		87			1.8
(88)	0 g/d of IF	1–170	54		30.7	28			58	86	
	500 g/d of IF		54		32.1**	44			76	74	
(18, 56)	0% IF	5–100	23	23.6	36.5	33		57	59		
	5% IF		23	22.1	37.8	75		30	44		
(35)	0% IF	<30 to ≤ 150	138			43			86	96	1.96
	2% IF		115			59**			93*	92	1.57**
(90)	0% IF	1–120	48	20.3	+1.7	42		62		149	2.9
	2.6% IF		51	20.2		39		82**		115**	2.4**
(89)		1–120	Primiparous								
	0% IF		19	20.6	26.4	74**		84			
	2.5% IF		21	20.3	30.9**	33		76			
			Multiparous								
	0% IF		29		32.4	42		69			
	2.5% IF		33		36.6**	33		51			
(38)	0% IF	1–120	21	19.5	24.8	33		52		76	1.35
	2.2% IF		22	19.4	26.4	45		86**		84	1.45
(86)	0% IF	1–180–200	223		+1.0 for heifers +1.5 for cows	49		85	93	138	1.74
	450 g/d of IF		220			46		79	98**	146	1.71
(33, 68)	0% IF	15–98	20	18.5	32.5	44		45			1.2
	3% IF		20	17.9	34.8**	12		10**			0.5
(83)	0% PHT[3]	1–151	16	19.9	32.0			94		88	1.36
	2% PHT		16	20.6	33.2			94		95	1.25
(93)	0% Tallow	15–84	34	24.6	30.7	33		44			
	3% Tallow		34	23.4**	31.4	44		62*			
						Bunk	Calan				
(17)	0% FM[2]	12–125	31	20.4	32.7	68	67	84		87	1.2
	3.5% FM		31	20.5	33.8	89*	33**	86		82	1.4
(12)	0% FM		67	22.2				52	43		
	7.3% FM	1–112	65	20.5				72**	52		
(2)	0% FM		41		31.6				44	107	2.31
	800 g/d of FM		39		30.9				64**	94	1.62**
(14)	0% FM	25–113	166	23.6	42.2	42		65	66	78	1.5
	2.7% FM		175	24.0	42.6	41		60	70	79	1.4
	0% FM	23–105	146	24.7	46.1	20		32	33	74	1.4
	2.8% FM		154	25.0	46.2	22		41*	41	77	1.4

[1]SB = Soybeans, WCS = whole cottonseed, IF = inert fat, PHT = partially hydrogenated tallow, and FM = fish meal.

*$P < 0.10$.

**$P < 0.05$.

TABLE 3. Effect of supplemental fat on numbers of follicle size classes from lactating dairy and beef cows.

Reference	Fat source	Follicle size class 3–5 mm	6–9 mm	10–14 mm	≥15 mm
		(mm)			
(104)	Cottonseeds	*	*		
(82)	Soybean oil		*		
(101)	Soybean oil and tallow		*		
(9)	Tallow and yellow grease				*
(47)	Ca-LCFA[1]			*	
(67)	Ca-LCFA	*	*		*

[1]Calcium salts of long-chain fatty acids.

*Increase from fat supplementation ($P < 0.06$).

SUPPLEMENTAL FAT INFLUENCES REPRODUCTIVE PERFORMANCE

Has the reproductive performance of lactating dairy cows been improved by the inclusion of supplemental fat in the diet? The results are mixed. Of the studies reporting conception or pregnancy rate data (Table 2), 11 studies (2, 12, 14, 17, 35, 38, 85, 86, 88, 90, 93) reported an improvement ($P < 0.10$ or ≥15 percentage unit difference between means) either in first AI service conception rate or in the overall rate of conception or pregnancy. Mean improvement in rate of conception or pregnancy of studies reporting a positive response was 17 percentage units. Fat sources stimulatory to reproduction included Ca soaps of long-chain fatty acids (**Ca-LCFA**) (n = 5), fish meal (n = 4), tallow (n = 1), and prilled fat (Dairy Fat Prills; BP Nutrition, Ltd., Wincham, Norwich, Cheshire, United Kingdom) (n = 1). In addition to improved conception rate, one large study (n = 443) from five herds in Wisconsin reported other positive benefits (86). A greater proportion of cows fed Ca-LCFA showed stronger signs of estrus (71.4 vs. 65.6% exhibited standing estrus), had more active ovaries (75.4 vs. 69.5%, as determined by rectal palpation done every 2 to 4 wk), and required less exogenous $PGF_{2\alpha}$ to induce estrus (43.7 vs. 55.7%).

Three studies reported a strong negative influence of feeding fat on conception rate at first AI service. In each case, a lowered conception rate was accompanied by a large increase in milk production. Primiparous cows fed Ca-LCFA produced 4.5 kg/d more 3.5% FCM, took longer to regain lost BW, and had lower conception rates at first AI service (73.7 vs. 33.3%) than did control cows (89). The gap between treatments was narrowed by 120 d PP (84 vs. 76%) but still numerically favored control cows. In an Illinois study, production of FCM increased 2.3 kg/d (33), but pregnancy rate (68) decreased from 45 to 10% when Ca-LCFA was fed. In a third study, production of milk was increased 3.8 kg/d, and conception rate at first AI decreased from 67 to 33% when fish meal (3.5% of diet DM) partially replaced soybean meal in diets for Holstein cows (n = 18) fed from Calan gates (American Calan, Inc., Northwood, NH), but conception rate increased from 68 to 89% for cows fed from open bunk feeders (n = 44) (17). By 150 d PP, pregnancy rates did not differ according to dietary treatment. Lowered conception rate may have resulted from the slow adaptation to Calan gate (American Calan, Inc., Northwood, NH) feeders by cows fed fish meal (17). A significant stimulation to milk production by fat supplementation appeared to be antagonistic to fertility. High milk production has been linked to lowered fertility in lactating dairy cows but is inferior to energy status (**ES**) as an influence on reproductive performance. The effect of fat supplementation on ES is discussed in a subsequent section.

The number of days open was unaffected by fat supplementation with one positive exception (90). However, the number of AI per conception was decreased in three studies (2, 35, 90) by feeding a diet fortified with fat (Table 2).

The inclusion of some fat sources into the ration at 2 to 3% of dietary DM within the first 30 d PP has resulted in dramatic improvements in the rates of conception or pregnancy in over half of the studies cited in Table 2.

TABLE 4. Effect of fat supplementation on diameter of dominant ovarian follicles of dairy cows or heifers.

Reference	Fat source	Experimental diet Control	Fat
		(mm)	
(67)	0% vs. 2.2% of Diet DM as Ca-LCFA	12.4[a]	18.2[b]
(65)	0 ml vs. 200 ml of Soybean oil infused	7.0[c]	10.2[d]
(64)	0% vs. 2.2% of Diet DM as Ca-LCFA	16.1[c]	18.7[d]
(75)	0.45 kg of Tallow vs. 0.45 kg of yellow grease infused	16.9[a]	20.9[b]
(9)	0 vs. 1.9% of Diet DM as tallow and yellow grease	11.0[a]	13.5[b]
Mean		12.7	16.3

[a,b]Means in the same row with no common superscript letter differ ($P < 0.15$).

[c,d]Means in the same row with no common superscript letter differ ($P < 0.10$).

DIETARY FAT INFLUENCES OVARIAN FOLLICLES

Not only has fat supplementation improved the conception of lactating dairy cows in many studies, but the development of ovarian follicles during the early PP period also has been stimulated fairly consistently (Tables 3 and 4).

Corn was replaced with Ca-LCFA at 2.2% of dietary DM in a diet containing 14.5% whole cottonseed and fed to dairy cows starting at parturition (67). Using ultrasonography, the number of medium-sized follicles (6 to 9 mm) increased, and the number of small follicles (3 to 5 mm) decreased, prior to 25 d PP in cows fed additional fat. These changes may reflect an increased movement of small follicles into the medium size class. During a synchronized estrous cycle, initiated after 25 d PP, the number of small (3 to 5 mm) and large (>15 mm) follicles increased in cows fed Ca-LCFA. A greater number of small follicles may reflect a greater pool of follicles available for subsequent development. The diameters of the largest (18.2 vs. 12.4 mm) and second largest (10.9 vs. 7.4 mm) follicles were greater in cows fed Ca-LCFA during the synchronized estrous cycle.

This increase in follicle size from dietary Ca-LCFA was repeated in a second study (64) and was shown to be due to the fat source itself rather than to a shift toward a more positive ES. Lactating Holstein cows consuming Ca-LCFA at 2.2% of dietary DM had a larger second wave dominant follicle than did cows fed a diet of similar energy density but devoid of Ca-LCFA (16.1 vs. 18.7 mm). Cows were of similar ES.

Dairy cows were fed a blend of tallow and yellow grease (88:12, wt/wt) at 0, 2.2, or 4.4% of dietary DM from d 0 to 84 PP (9). The number of large ovarian follicles (>15 mm in diameter) was about four times greater on d 14 of the estrous cycle in cows fed supplemental fat than in control cows. In addition, the diameter of the largest follicle from d 8 to 14 of the estrous cycle was greater in cows fed the 2.2% than in cows fed the 0% fat diet (13.5 vs. 11.0 mm). Fat also stimulated follicle size when only cows that ovulated the first dominant follicle PP were considered. From this group of cows, the mean diameter of the largest follicle was 9.8, 13.9, and 12.0 mm for cows fed increasing amounts of fat. Cows fed fat at 2.2% of dietary DM experienced a shorter interval to first ovulation than did the other two dietary groups (21.4 vs. 45.3 and 37.4 d PP).

Holstein cows, fed diets of either 0 or 2.2% Ca-LCFA starting at calving, were examined weekly by rectal palpation for the first 60 d PP (38). The accumulated ovarian volume on the ipsilateral side of the corpus luteum (**CL**) compared with the contralateral side was less, as expected, for control cows (365 vs. 619 mm^3); the volume difference for the lipid-supplemented cows was less (450 vs. 567 mm^3; fat by side interaction). The number of CL (0.85 vs. 1.05) and the size of the largest CL (12.2 vs. 17.2 mm) tended to be greater in cows fed Ca-LCFA.

Four ruminally cannulated lactating dairy cows arranged in a 4×4 Latin square design were infused abomasally with 1) water, 2) 1 kg/d of glucose, 3) 0.45 kg/d of tallow, and 4) 0.45 kg/d of yellow grease (75). Estrous cycles were synchronized at the beginning of each period of the Latin square. The first wave dominant follicle of cows infused with yellow grease grew faster (1.4 vs. 0.5 mm/d) and was larger in diameter (20.9 vs. 16.9 mm) than that of cows infused with tallow (2% linoleic acid) as measured using ultrasonography. Growth and size of the second wave dominant follicle were unaffected by fat source.

This effect of fat upon the number and size of ovarian follicles has been confirmed using lactating beef cows. Lactating Simmental cows were supplemented with 1 kg/d of Ca-LCFA, but energy intake was equalized with the control diet by adjusting the amount of DM fed (47). The numbers of 10- to 15-mm size follicles measured at approximately 20 to 25 d PP were increased (0.25 vs. 0.92) in cows fed Ca-LCFA. Whole cottonseeds also have proved to be effective. The feeding of whole cottonseeds at 30% of diet DM to lactating, crossbred Brahman cows resulted in a greater number of medium-size follicles (3.1 to 9.9 mm) at 19 to 21 d PP as measured by ovariectomy compared with those of cows fed a diet similar in metabolizable energy (104). Cycling beef heifers fed soybean oil or animal tallow had more medium-sized follicles than did cows fed an isoenergetic control diet (101). Sexually mature beef heifers fed soybean oil at 5.4% of dietary DM had more medium-sized follicles (5 to 9.9 mm diameter) in the estrous cycle before initiation of superovulation than did cows fed a diet similar in metabolizable energy but lacking soybean oil (82).

Delivery of fats for metabolism by routes other than feeding have influenced follicle development as well. Holstein heifers (BW = 310 kg) were infused intravenously with either Intralipid® (20% soybean oil emulsion and 50% of fatty acids as linoleic acid; Kabivitrum Inc., Alameda, CA) or physiological saline on d 9 to 13 of a synchronized estrous cycle (65). On d 16 of the cycle, the mean number of ovarian follicles >3 mm in diameter was increased threefold from 2 to 6, and the diameter of the largest follicle was increased from 7.0 to 10.2 mm in heifers infused with soybean oil emulsion.

Three studies reported negative effects of feeding ruminally inert fat on ovarian activity. Through 200 d PP, a greater proportion of lactating dairy cows (n = 443) fed Ca-LCFA than cows not fed Ca-LCFA were anestrus (8.6 vs. 19.2%) (86). This result was due primarily to a greater incidence rate of anestrus among the primiparous cows (10.5 vs. 26.9%). In a second study, 9 of 16 cows were diagnosed with cystic ovaries during 151 d of feeding partially hydrogenated tallow (Alifet®; Alifet USA, Inc., Cincinnati, OH); only 4 of 16 cows fed control diets were diagnosed with cystic ovaries (83). Finally, more cows (7 of 23) fed prilled fat than control cows (1 of 23) were not inseminated in the first 100 d PP. Of the cows fed fat, 5 of the 7 cows that were not bred were not detected in estrus during the experiment (18).

The impact of the larger ovarian follicles on conception rate has not been defined. Follicle size may have no relationship to secretion of estradiol or subsequent secretion of progesterone by the CL. Perhaps a greater frequency of follicles or a larger dominant follicle at the time of pregnancy recognition (d 17 post-AI) might be expected to antagonize CL maintenance and contribute to a lower pregnancy rate. However, this situation appears not to be the case because conception rates often are improved by fat supplementation. Follicles that ovulated in the first follicular wave PP were of greater diameter (20 vs. 16 mm) than those that failed to ovulate (9). Cystic follicles are very large persistent follicles (>25 mm) that fail to ovulate. Only one study reported greater occurrence of cystic follicles when fat was fed (83).

POSSIBLE MEANS BY WHICH DIETARY FAT INFLUENCES REPRODUCTIVE PERFORMANCE

The mechanism or mechanisms by which dietary fat improves reproductive performance has not been elucidated. Several hypotheses have been proposed: 1) an amelioration of a negative ES, thus leading to an earlier return to estrus PP and, therefore, improved fertility; 2) an increase in steroidogenesis favorable to improved fertility; 3) manipulation of insulin so as to stimulate ovarian follicle development; and 4) a stimulation or inhibition of the production and release of $PGF_{2\alpha}$, which influences the persistence of the CL.

Effect on ES

An additional objective of dietary supplemental fat in the early PP period is to lessen the negative ES of the herd. The energy density of the diet is increased by replacing grain or forage with fat. However, the daily energy intake may remain unchanged because of lowered DMI of fat-supplemented diets (1, 45, 56, 80). An increase in endogenous cholecystokinin may be responsible for lowered DMI of cows fed supplemental fat (22). In addition, production of milk or FCM often is increased when fat is supplemented. These increases likely were due to an increased delivery of metabolizable energy when fat was supplemented (1, 80).

Most studies report that the feeding of supplemental fat in the form of ruminally inert fat (9, 56, 64, 95), inert fat plus whole cottonseed (45), whole cottonseed (26), or tallow and yellow grease (9) during the early PP period did not influence the ES of the cows, often because of a nonsignificant depression in DMI by cows fed the supplemental fats. However, a beneficial effect of supplemental dietary fat on ES of lactating dairy cows has been reported using whole cottonseed (45), tallow plus Ca-LCFA (79), and partially hydrogenated tallow and lard (10). However, supplemental fat sometimes has had a detrimental effect on ES. The negative ES during the early PP period was made worse by the dietary tallow, again because of lowered DMI (93). Others, using changing BW or body condition score (**BCS**) as evidence of a more negative or positive ES, reported either no change in ES (83) or a more negative ES (89, 90) for cows fed supplemental fat than for cows in the control group. Other reasons for lowered DMI include lowered acceptability of the fat-supplemented diet, increased rumen fill from reduced fermentation, and chemostatic mechanisms to maintain relatively constant intake of energy.

If the discussion is narrowed to include only those studies that reported an improved rate of conception and pregnancy because of fat supplementation (Table 2), was the improved ES responsible for the improvement in conception or pregnancy rates? Unfortunately, the ES of experimental cows was calculated in only two studies (17, 93). Dairy cows fed tallow at 3% of dietary DM had a greater pregnancy rate than did control cows despite a more negative calculated mean net ES from wk 2 to 12 PP (93). Conception rate at first AI decreased from 67 to 33% when fish meal replaced soybean meal in diets fed to cows via Calan gates with no change in mean ES (17). If a change in BW or BCS is used as an indicator of ES, cows experiencing improved conception rates did so either without an improvement in BW or BCS (2, 17, 38) or despite a worsening BW or BCS (14, 90). Improved BW did match improved conception rates of cows fed fat in two studies (12, 85). Also, cows fed

Ca-LCFA lost more BW for the first 35 d PP than did control cows before reversing and gaining more BW for the duration of the study (88). Ferguson et al. (35) did not report BW change for herds in Pennsylvania that were mostly responsible for the improvement in conception rate with dietary Ca-LCFA. Although evidence exists that dietary fat can improve the ES of cattle, the improvement in reproductive performance occurred in several instances apart from an improving ES of the experimental cattle.

There is a strong need for more studies in which ES is determined. However, because large numbers of cows are required to test adequately for dietary effects on conception and pregnancy rates, commercial dairy farms are a natural location for these studies. A limitation to this approach, however, is that DMI for individual cows cannot be determined; therefore, ES cannot be a dependent variable for statistical analysis.

Effects on Steroidogenesis

Endocrine effects on follicular development is still very much a fertile area for research. What hormones are responsible for the recruitment and growth of ovarian follicles and development of the CL? Is the production and secretion of these hormones stimulated by an additional supply of fatty acids for metabolism?

Cholesterol. The concentration of plasma cholesterol is increased consistently under regimens of supplemental fat (41). Lipoprotein cholesterol is increased by dietary fat supplementation (40). This increased cholesterol likely is due to the need for increased absorption of fatty acids packaged in the chylomicrons and very low density lipoproteins from the small intestine. Most (90 to 95%) cholesterol in bovine blood is found in the high density lipoproteins (**HDL**) (40). Cows fed ruminally inert, highly saturated fat had 21% greater concentrations of plasma HDL cholesterol than did control cows (15). Not only are blood concentrations increased, but follicular fluid concentrations have been increased during fat supplementation. Beef cows that were supplemented with soybean oil (5.4% of diet DM) had a greater concentration of total cholesterol in serum and HDL cholesterol in the follicular fluid of a surgically removed ovary than did control cows (82). Whole cottonseeds in the diet also increased the concentration of HDL in follicular fluid of beef cows (104). The fat content of luteal cells was increased by fat supplementation. Electron microscopic examination of slices of CL tissue revealed that lipid occupied a greater percentage of cell area in luteal cells from beef heifers fed Ca-LCFA from 100 d prepartum to their third estrous cycle PP than in cells from cows that were not fed Ca-LCFA (46). Cholesterol can be supplied from circulating lipoproteins, synthesized de novo by luteal cells from acetate, and synthesized from intracellular storage of cholesterol esters. Most of the cholesterol in the ovary comes from the direct transport of lipoprotein cholesterol from the blood (73).

Cholesterol serves as a precursor for the synthesis of progesterone by ovarian luteal cells. High density lipoproteins and low density lipoproteins demonstrated a similar ability to synthesize progesterone in vitro. In addition, cholesterol uptake by luteal cells may be independent of receptors (16). Secretion of progesterone is the main function of the CL. Progesterone not only prepares the uterus for implantation of the embryo but also helps maintain pregnancy by providing nourishment to the conceptus. Between 25 and 55% of mammalian embryos die in early gestation; many of these losses are due to inadequate function of luteal cells (73). Increased concentrations of plasma progesterone have been associated with improved conception rates of lactating ruminants. Similarly, progesterone concentration prior to AI has been associated with greater fertility. In a field study involving 426 lactating dairy cows, blood was sampled on 58 ± 3 d PP for multiparous cows and 72 ± 3 d for primiparous cows and then analyzed for progesterone. Cows were bred approximately 3 d later in a synchronized estrus scheme. Conception rate increased 1.44% for every 1 ng/ml increase in plasma progesterone (R^2 = 0.11) (96). The recovery of embryos 7 d after estrus increased as plasma progesterone concentration increased just prior to AI (11). In either association, dietary fat, which stimulates ovarian cyclicity or CL function, would contribute to increased fertility.

Ruminants fed or infused with supplemental fat often have small increases in concentrations of progesterone in blood (Table 5). Studies reporting this effect include beef cattle fed whole sunflower seeds (99), whole cottonseeds (106), and Ca-LCFA (46, 47); lactating dairy cows fed tallow (93), Ca-LCFA (38, 64, 90, 95), and prilled fatty acids (18); and ewes infused intravenously with soybean oil or olive oil (13). The concentrations of progesterone in follicular fluid also was higher for beef cows that were fed soybean oil at 5.4% of dietary DM than for control cows (82). Increased progesterone suggests that luteal function is enhanced by dietary fat.

From these studies, the implication is that the additional circulating concentrations of cholesterol

TABLE 5. Effect of fat supplementation on concentration of plasma progesterone of lactating dairy cows.

Reference	Time of measurement	Diet		SEM
		Control	Fat	
		(ng/ml)		
(64)	1–12 d of estrous cycle	4.2[a]	5.2[b]	0.8
(18)	9–15 d of estrous cycle	6.6[a]	7.7[b]	0.3
(90)	8–20 d of estrous cycle	Greater accumulation[a,b]		
(95)	5–12 wk PP[1]	4.5[a]	6.0[b]	0.5
(38)	1–7 wk PP	Greater accumulation[a,b]		
(93)	2–12 wk PP	4.2[a]	4.8[b]	0.3

[a,b]Means within the same row with no common superscript letter differ ($P < 0.05$).

[1]Postpartum.

stimulated by the feeding of fat increases the synthesis of progesterone by follicular and luteal cells. However, Carroll et al. (16) reported that maximum in vitro synthesis of progesterone by bovine luteal cells occurred at much lower concentrations of HDL than those found in plasma. Those researchers suggested, therefore, that lipoprotein cholesterol should not be limiting for the synthesis of progesterone. In addition, the cholesterol to protein ratio in lipoproteins did not change when supplemental fat was fed (15). Therefore, the in vitro synthesis of progesterone by luteal cells incubated with lipoproteins was similar for cows fed diets of 0 or 7% supplemental fat (15).

The fatty acid profile of the dietary fat may influence the propensity of animals to increase plasma progesterone. Mature ewes were infused intravenously with saline, soybean oil, or olive oil for 5 h on d 9 through 13 of an estrous cycle (13). Serum cholesterol was increased by fat infusates, and olive oil was more effective than soybean oil (127, 141, and 153 mg/dl for saline, soybean oil, and olive oil, respectively). However, soybean oil infusion resulted in a greater progesterone response than did infusion of olive oil at 2.5 h postinfusion. Therefore, the greatest concentration of serum cholesterol did not coincide with the greatest concentration of serum progesterone. Greater concentrations of linoleic and linolenic acids in soybean oil may depress $PGF_{2\alpha}$ to a greater extent, thus allowing greater synthesis and secretion of progesterone from granulosa and luteal cells.

Recent work by Hawkins et al. (46) suggests that increases in plasma progesterone in cows fed fat-supplemented diets may not be due to increased synthesis but rather to reduced clearance of progesterone from circulation. Beef heifers were fed either 0 or 0.57 kg/d of Ca-LCFA from 100 d prepartum through the third estrous cycle PP. Mean plasma cholesterol and progesterone concentrations were increased based upon blood samples collected every other day. On d 12 to 13 of the third cycle, heifers were ovariectomized. Repeated blood samples that were taken immediately before and after ovariectomy indicated that the half-life of serum progesterone was increased in heifers fed fat. Based on several assumptions, an estimated metabolic clearance rate for progesterone was calculated for both treatment groups. The prolonged elevation in concentration of progesterone was thought to be due to a reduced rate of clearance from the blood rather than to an increased rate of secretion (46).

LH. Luteinization (morphological and biochemical changes) of theca and granulosa cells of the developing follicle result from the preovulatory surge of LH. After ovulation, granulosa cells differentiate into luteal cells of the CL that are capable of secreting progesterone after exposure to LH (29). Receptors for lipoproteins develop on the luteal cells in order to take up cholesterol for the synthesis of progesterone. Receptors also develop on the developing CL for LH. Nursing Simmental cows fed 1 kg/d of Ca-LCFA tended to have greater mean concentrations of serum LH (1.12 vs. 1.47 ng/ml) and enhanced follicle growth than did cows that were not fed Ca-LCFA (47). Plasma LH (mean concentration, number of pulses, or pulse amplitude) was not affected during the luteal phase of the estrous cycle by dietary Ca-LCFA (89). However, during the follicular phase, mean plasma concentrations of LH were increased in primiparous cows but decreased in multiparous cows when Ca-LCFA were fed. Despite higher LH, primiparous cows fed fat had lower conception rates at first AI. Progesterone concentrations in plasma were unaffected during this same time period. On d 10 PP, the LH profile (mean concentration, baseline concentration, number of peaks in 8 h, and peak amplitude) of dairy cows was unaffected by dietary Ca-LCFA (67). However, as LH pulse amplitude increased, the diameter of the largest follicle increased, and ES was less negative. Insufficient evidence exists to conclude that increased plasma progesterone results from greater differentiation of granulosa cells to luteal cells or greater stimulation of luteal cells during diestrus by enhanced LH secretion.

Effects on Insulin

Insulin has proved to be a powerful stimulator of ovarian follicle cell function. Addition of insulin to granulosa cells that were collected from small (1 to 5 mm) and large (>8 mm) follicles from ovaries of

slaughtered beef and dairy cows stimulated greater cell proliferation and production of progesterone than was found with cells to which no insulin had been added (62, 94).

However, the effect of fat supplementation on plasma concentrations of insulin are mixed. A plot of insulin concentrations of control cows versus insulin concentrations supplemented cows from 17 studies appears in Figure 1. Data points represented by a triangle were nonsignificant changes in insulin; data points represented by a circle were significant changes caused by fat supplementation. In 8 studies, plasma insulin was depressed significantly in lactating dairy cows supplemented with fat. Insulin concentrations usually reflect energy intake. Concentrations of plasma insulin increased gradually as days PP increased and as DMI of dairy cows fed six different diets also increased (66). Differences in plasma insulin that were due to diet and day were eliminated when ES was used as a covariate in the statistical model, suggesting that insulin differences among diets were due to differences in ES (66). In studies in which fat supplementation improves ES, plasma insulin concentrations may be increased apart from the direct effect of fat (10).

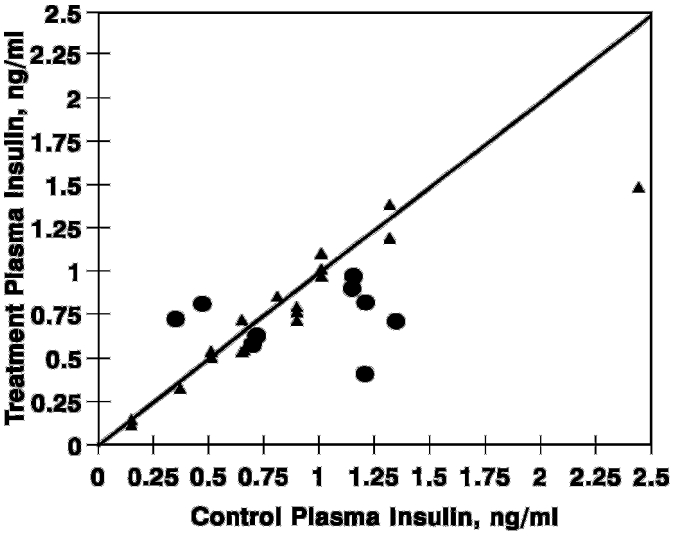

Figure 1. Effect of supplemental dietary fat on blood or plasma concentrations of dairy cattle. Points above the diagonal line represent studies in which supplemental fat numerically increased blood insulin concentrations, and points below the diagonal line represent studies in which supplemental fat numerically decreased blood insulin concentrations. Triangles represent nonsignificant effects, and circles represent effects ($P < 0.10$) from fat supplementation. Data are adapted from studies of Ballantine and Herbein (4), Beam and Butler (9), Blum et al. (10), Chilliard and Ottou (20), Choi and Palmquist (21), Cummins and Sartin (26), Gagliostro et al. (37), Garcia-Bojalil (38), Horner et al. (49), Lough et al. (63), Lucy et al. (66, 64), Mohamed et al. (70), Oldick et al. (75), Palmquist and Moser (78), Schneider et al. (85), and Smith et al. (91).

If the production or release of insulin is depressed by dietary fat supplementation, then the resulting influence on follicle development might be expected to be negative. However, the lower plasma concentration of insulin might result from a greater clearance of insulin from the blood by tissues, including those of the reproductive tract, which would in turn stimulate the growth of ovarian follicles. If production of insulin is indeed depressed, how might this benefit the development of follicles or reproductive performance? Spicer et al. (94) reported that bovine granulosa cells tended to produce less IGF-I when cultured with insulin and GH. Because IGF-I is a potent stimulator of bovine granulosa cells in vitro (94), suppression of insulin through the feeding of fat may allow IGF-I to affect follicle development positively. More studies are required to assess the effect of circulating insulin concentrations on follicle development in vivo.

Lower blood insulin also is known to enhance lipolysis in adipose tissue of the lactating dairy cow. Evidence summarized by Grummer and Carroll (41) suggests that net triglyceride hydrolysis in adipose tissue is increased when additional fat is added to the diet. Adipose tissue collected from sheep and steers released more fatty acids in vitro when protected fat was included in the diet (108). Lipolytic activity was greater from adipose tissue collected from lactating cows that had been duodenally infused with rapeseed oil (36), and greater lipolytic activity can result in greater loss of BW or body condition. Studies (89, 90) have reported greater BW loss by cows fed supplemental fat. At 60, 120, and 240 d PP, adipocytes were collected via biopsy from Holstein cows fed 1) no supplemental fat, 2) whole cottonseeds at 12% of diet DM, or 3) whole cottonseeds plus Ca-LCFA at 2.7% of diet DM. At all days PP, adipocytes from cows fed supplemental fat were less lipogenic when incubated with acetate than were adipocytes from cows not supplemented with fat (69). Thus, the consumption of supplemental dietary fat reduces lipogenesis by adipose tissue. Increased lipolysis results in increased concentrations of plasma NEFA. Concentrations of plasma NEFA nearly always are elevated in cows fed supplemental fat (41).

The mammary gland can take up NEFA from plasma (39), although chylomicrons and very low density lipoproteins are the major sources of fatty acids in the mammary gland (71). De novo synthesis of fatty acids by the mammary gland was reduced when additional fatty acids were supplied from the blood (39). The synthesis of fatty acids by the mam-

mary gland requires NADPH, a large part of which is supplied during the oxidation of glucose in the pentose phosphate shunt (8). The lower demand for glucose by the mammary gland for triglyceride synthesis allows greater availability of glucose for energy metabolism in other tissues. If glucose is spared, this sparing effect is not evident from blood glucose concentrations. Blood glucose concentrations generally are not changed under conditions of supplemental fat (41). The mean difference in plasma glucose concentrations between cows fed control and fat-supplemented diets in 52 comparisons was 0 ± 3 mg/dl (19). Studies since 1993 confirm the lack of a consistent effect of dietary fat on blood glucose. Concentrations of plasma glucose were unaffected in lactating dairy cows fed high amounts of oil corn and tallow (23) or abomasally infused with saturated or unsaturated free long-chain fatty acids (24). Alternatively, plasma glucose was decreased in cows duodenally infused with 0.64 kg/d of rapeseed oil (20). At wk 7 PP, plasma glucose decreased from 72.7 to 68.7 g/dl for cows fed Ca-LCFA at 2.5% of diet DM but was unchanged at 13 wk PP (87). Tallow fed at 2% of diet DM from 1 to 120 d PP resulted in greater concentrations of glucose in plasma collected every 2 wk (6). Of course, plasma concentrations give no indication of possible differences in blood flow or clearance rates. Greater availability of glucose for metabolism may serve as a key metabolic signal for the release of gonadotropins for follicle development.

Cows that were duodenally infused with rapeseed oil (20) showed evidence of insulin resistance (defined as an inability of insulin to stimulate tissue glucose utilization [76]) compared with that of control cows; that is, the concentration of plasma glucose was greater at 45 to 90 min after an intravenous pulse-dose of glucose, an increased apparent half-life of injected glucose, and a greater concentration of plasma insulin after an insulin challenge that failed to decrease glycemia in cows infused with fat.

Effects on the Secretion of $PGF_{2\alpha}$

Prostaglandins play a wide role in mammalian physiology and metabolism. They are classified as belonging to different series based upon the nature and position of oxygen-containing substituents present in the cyclopentane ring. Most mammalian organs synthesize prostaglandins, although the activity of synthesis varies between organs. Prostaglandins are local regulators; therefore, they generally are synthesized in close proximity to the cells they influence. Within the reproductive tract of cows, uterine tissue is a primary source of the F series prostaglandins (e.g., $PGF_{2\alpha}$) during the early PP period (43). Concentrations of 13, 14-dihydro-15-keto-$PGF_{2\alpha}$ metabolite (**PGFM**) in plasma had risen dramatically to a peak of 1800 pg/ml by 3 to 4 d PP. This rise is associated with the regression of the CL of pregnancy and PP regression of the uterus. (The PGFM is produced as the uterus and lung metabolize $PGF_{2\alpha}$). Over the next 2 wk, PGFM gradually returned to baseline concentrations. The uterus then releases $PGF_{2\alpha}$ regularly over the following weeks to regress each newly formed CL in order to initiate a new estrous cycle if the cow is not pregnant. If the cow does conceive, $PGF_{2\alpha}$ release from the uterus is inhibited in order to preserve the CL on the ovary and maintain pregnancy. Because $PGF_{2\alpha}$ has an effect on the regression of the CL, concentrations of plasma progesterone are related inversely to $PGF_{2\alpha}$ concentrations during CL regression in late diestrus. However, progesterone priming of the uterus is essential to induce uterine lipids for the subsequent synthesis of PGF_{α}.

In summary, prostaglandins have an important function in reestablishing estrous cycles both immediately after parturition and thereafter until conception occurs. Upon conception, $PGF_{2\alpha}$ must be prevented from regressing the CL in order to maintain pregnancy (e.g., to prevent early embryonic death).

Some polyunsaturated, long-chain fatty acids can serve as substrates for biosynthesis of the prostaglandins and as inhibitors of the prostaglandins.

Fatty acids as substrates. Twenty-carbon polyunsaturated fatty acids, including dihomo-γ-linolenic acid ($C_{20:3}$), arachidonic acid ($C_{20:4}$), and eicosapentaenoic acid ($C_{20:5}$), act as substrate for synthesis of the one, two, and three series prostaglandins, respectively. *Cis*-linoleic acid ($C_{18:2}$), which is commonly found in natural fat sources, can be desaturated and elongated to form dihomo-γ-linolenic acid ($C_{20:3}$), which serves as an immediate precursor for the series 1 prostaglandins or can be desaturated further to arachidonic acid ($C_{20:4}$), which serves as an immediate precursor for the series 2 prostaglandins of which $PGF_{2\alpha}$ is a key member. For mammals, arachidonic acid is the main substrate for prostaglandins. α-Linolenic acid ($C_{18:3}$) can be desaturated and elongated to form eicosapentaenoic acid ($C_{20:5}$), which serves as an immediate precursor for the series 3 prostaglandins. One regulatory enzyme in the synthesis of prostaglandins from fatty acids is Δ-6-desaturase for the conversion of 1) *cis*-linoleic to dihomo-γ-linolenic (44) and 2) α-linolenic to eicosapentaenoic acid. A second regulatory enzyme is

cyclooxygenase for the conversion of 1) dihomo-γ-linolenic acid to the series 1 prostaglandins and 2) arachidonic acid to the series 2 prostaglandins. Cyclooxygenase and peroxidase enzymes are part of the prostaglandin endoperoxide synthase complex, which catalyzes the synthesis of prostaglandins from 20-carbon fatty acids.

Fatty acids as inhibitors. Although the synthesis of prostaglandins depends upon the supply of fatty acids, these fatty acids also can inhibit prostaglandin synthesis. Linoleic acid has demonstrated inhibitory effects both in vivo and in vitro. Supplementation of linoleic acid has reduced the production of arachidonic acid in preweaned calves (52). Using in vitro incubation techniques, additional linoleic acid reduced the production of $PGF_{2\alpha}$ by bovine pulmonary artery endothelial cells (58) and by oral squamous carcinoma cells (32). In addition, linoleic acid can be converted to a shunt metabolite, eicosadienoic acid ($C_{20:2}$), rather than to arachidonic acid (58) when excess linoleic acid is present, thereby reducing synthesis of series 1 and 2 prostaglandins. Linoleic acid is an inhibitor of prostaglandin synthesis that is produced by the endometrium in response to the conceptus in order to preserve its integrity (27, 100). The inhibition mechanism is thought to occur by the competition of linoleic acid with arachidonic acid for binding of the key enzyme, cyclooxygenase. Arachidonic, eicosapentaenoic, and docosahexanoic ($C_{22:6}$) acids also have been shown to inhibit cyclooxygenase activity (92). Activity of Δ-6-desaturase also can be inhibited to reduce synthesis of $PGF_{2\alpha}$. α-Linolenic acid can compete with *cis*-linoleic acid for Δ-6-desaturase to the extent that eicosapentaenoic acid is produced rather than arachidonic acid (44), thus, potentially depressing synthesis of $PGF_{2\alpha}$. High concentrations of 20-carbon fatty acids other than arachidonic acid (i.e., $C_{20:3}$ and $C_{20:5}$) can compete with arachidonic acid for active sites of prostaglandin-endoperoxide synthase complex, therefore, reducing the conversion of arachidonic acid to the series 2 prostaglandins (103).

The amount of particular fatty acids reaching the target tissues likely influence whether prostaglandin synthesis is stimulated or inhibited.

Concentrations of plasma PGFM postinjection of oxytocin on d 15 of synchronized estrous cycles were reduced greatly in lactating dairy cows that were abomasally infused with 0.45 kg/d of yellow grease compared with infusions of tallow, glucose, and water (75). Yellow grease and tallow differed in the proportion of many fatty acids, and this suppressing effect on PGFM cannot be attributed to only one fatty acid. However, linoleic acid is a prime candidate of influence because yellow grease supplied 78 g/d of linoleic acid, but tallow supplied only 9 g/d.

In a study using ewes, concentrations of serum PGFM were increased by the intravenous infusion of soybean oil or olive oil compared with the effects of saline infusion (13). Olive oil stimulated a greater response than did soybean oil at 5 and 8 h after initiation of infusion. Concentration of prostaglandin E_2 also was stimulated by olive oil but not by soybean oil. The length of the estrous cycle of ewes infused with olive oil was shortened by 1 to 1.8 d. Soybean oil contained more linoleic acid (50 vs. 8%) and linolenic acid (9 vs. 1%) than did olive oil. These fatty acids have been shown to inhibit conversion of arachidonic acid to the prostaglandins. A small amount of prostaglandin precursor (such as that found in olive oil) delivered to ovarian tissues may stimulate synthesis of $PGF_{2\alpha}$, thus, causing an earlier regression of the CL and a shortened estrous cycle. Soybean oil would have delivered much more linoleic and linolenic acid, thus, possibly preventing additional $PGF_{2\alpha}$ synthesis or release and not affecting the duration of the estrous cycle. Following injection of $PGF_{2\alpha}$ on d 16, regression of the CL in cows infused with yellow grease tended to occur more than 1 d later than it did in cows fed tallow (d 19.3 vs. 18.1 of the estrous cycle).

Whole cottonseed added to the diets of lactating beef cows doubled the average lifespan of a GnRH-induced CL compared with cows fed an isocaloric diet (7.2 vs. 15.3 d) (106). Prostaglandin concentrations were not measured, but a lipid-induced suppression of prostaglandin is compatible with a longer life of the CL.

As stated earlier, eicosapentanoic acid can compete with arachidonic acid for active sites of prostaglandin endoperoxide synthase, therefore, reducing the conversion of arachidonic acid to the series 2 prostaglandins (103). Eicosapentaenoic acid occurs naturally in fish meal along with docosahexaenoic acid ($C_{22:6}$) and constitute 11.6 and 6.6%, respectively, of the fatty acids in menhaden fish meal (8.4% ether extract). These fatty acids appear largely to escape biohydrogenation in the rumen (3, 77).

Inclusion of fish meal in the diets of lactating dairy cows has often improved conception rates. The repression of $PGF_{2\alpha}$ by these fatty acids may account for the improved conception rates. Alternatively, reducing the amount fed of ruminally degradable protein by replacing soybean meal with fish meal may have alleviated negative effects of excess systemic ammonia on fertility. However fertility also was improved

when fish meal replaced other sources of undegradable intake protein and the content of undegradable intake protein of the diet was kept constant. Starting at approximately 25 d PP, menhaden fish meal replaced a mixture of corn gluten meal, fish meal, meat and bone meal, and blood meal in a TMR for lactating dairy cows such that the dietary contents of undegradable intake protein were similar (14). An injection of GnRH was given at 51 ± 3 d PP to start the process of recruiting a new follicle. Seven days later, $PGF_{2\alpha}$ was injected to help ovulate the new follicle and regress the existing CL. Blood samples were taken 2 d after injection of $PGF_{2\alpha}$ and were measured for progesterone. Cows fed fish meal tended to have greater concentrations of plasma progesterone than those cows fed other protein feedstuffs (1.3 vs. 0.6 ng/ml); more cows fed fish meal had progesterone concentrations >1 ng/ml than did cows fed the other protein feedstuffs (4 vs. 29%). Overall pregnancy rate at 120 d PP was increased from 32 to 41%.

Estradiol. Estrogen stimulates uterine secretion of $PGF_{2\alpha}$ (61, 100). Furthermore, estrogen can increase the sensitivity of the CL to $PGF_{2\alpha}$ (50), thus causing a more complete regression of the CL; lowered estradiol-17β may prevent premature regression of the CL and prevent early embryonic death. Cows infused in the abomasum with tallow or yellow grease had lower concentrations of plasma estradiol (2.42 vs. 3.81 pg/ml) on d 15 to 20 of a synchronized estrous cycle than did cows infused with glucose (75). Mean concentrations of serum estradiol-17β were lower (1.64 vs. 1.41 pg/ml) for Simmental cows fed Ca-LCFA during the early PP period (47). The concentrations of estradiol-17β were lower in the follicular fluid of beef cows fed soybean oil at 5.4% of diet DM (82). When those granulosa cells were incubated with HDL, production of estradiol-17β by cells from cows fed unsupplemented diets was greater than that by cells from cows fed diets not supplemented with lipid (219 vs. 105 ng/ml). Other studies reported no effect of dietary fat on blood estradiol. Plasma estradiol was unchanged by supplemental Ca-LCFA (64, 89) during luteal and follicular phases of the estrous cycle of lactating dairy cows. Seven blood samples that were taken prior to 40 d PP revealed no differences in estradiol concentrations between cows fed diets without Ca-LCFA and those fed diets with 2.2% Ca-LCFA (67). Although more studies need to be conducted, fat supplementation appears to depress estradiol-17β.

Proposed mechanism of action by which supplemental fat enhances reproductive performance. The CL is regulated by at least three tissues: 1) the anterior pituitary gland, 2) the uterus, and 3) the conceptus. The anterior pituitary gland secretes LH to stimulate the differentiation of granulosa cells to luteal cells, the uterus produces $PGF_{2\alpha}$ to regress the CL, and the conceptus signals endometrial tissue to produce a prostaglandin synthetase inhibitor to inhibit $PGF_{2\alpha}$ secretion by the uterus.

Evidence is growing to support the hypothesis that dietary fats can modulate the survival of the CL in order to enhance pregnancy (Figure 2). Looking first at the anterior pituitary gland, the stimulatory effect of dietary fats on LH secretion is not well documented. In some studies, LH dynamics were stimulated by fat supplementation (47, 89) but were unchanged (67) or decreased (89) in others. More work needs to be done in this area. The mechanism by which supplemental fat would stimulate LH release is not known unless a glucose-sparing effect occurs at the mammary gland, providing greater glucose to signal the hypothalamus-pituitary control system to secrete more LH. At the uterine level, polyunsaturated fatty acids (linoleic and eicosapentanoic), which are known to inhibit the secretion of $PGF_{2\alpha}$, can be delivered to the circulation in increasing amounts potentially to exert this effect. If the secretion of $PGF_{2\alpha}$ is inhibited, the synthesis of progesterone proceeds unabated. If cholesterol is limiting for synthesis of progesterone, the additional fat should overcome the deficiency. Large amounts of data support the greater presence of progesterone in the blood or granulosa cells when supplemental fat is fed. At the same time, estradiol secretion is suppressed by supplemental fat (again supported by cited studies) in order to make the CL less sensitive to any $PGF_{2\alpha}$ that may be present. The life of the CL is maintained on the ovary to enable the survival of the conceptus. Corpora lutea that stay on the ovary longer (75, 106), appear in greater number (38), and are of greater diameter fit this hypothesis (38). At the embryonic level, linoleic acid inhibits cyclooxygenase in endometrial tissue to decrease the synthesis of $PGF_{2\alpha}$.

The amount of fatty acid intake that is needed to achieve these effects is not known. Delivery of supplemental linoleic acid is thought to be less than 100 g/d (Table 1) and delivery of eicosapentanoic acid to be approximately 7 g/d (14, 17). The amount of unsaturated fatty acid appearing in the small intestine likely is influenced not only by the source of supplemental fat but also by the amount of DMI influencing passage rate of digesta, the amount of fat fed influencing ruminal fermentation, and the fiber content of diet influencing mastication of oil seeds and release rate of oil. Perhaps the amount of dietary

linoleic acid that is needed to exert an effect depends on the storage of that fatty acid in tissues. Storage of fatty acids and turnover of fatty acid pools in tissues of experimental ruminants potentially influence fat effects on reproduction. The fatty acid composition of the diet and the duration of its feeding to animals prior to their assignment to a newly initiated experiment involving supplemental fat may influence and complicate interpretation of results. Effects of yellow grease on PGFM release appeared to carry over to subsequent experimental periods (75). That is, once a cow received yellow grease in an experimental period, release of PGFM was attenuated in all future periods, regardless of the type of infusate. Effects of particular fatty acid intakes by cattle on the phospholipid pools of fatty acids have not been investigated. This influence of diet on the phospholipid pools of fatty acids may lead to carry-over effects because phospholipids can function as a storage site for essential fatty acids. How long the fatty acid profiles of phospholipids remain altered once supplemental fat intake is eliminated needs to be examined. Residual effects of phospholipid pools potentially influence synthesis of the prostaglandins. These factors as well as others yet unidentified may make the impact of dietary fat on reproductive tissues and performance less than consistent.

Further alterations in the manner in which lactating dairy cows are fed may allow greater delivery of unsaturated fatty acids to the lower gut. Dietary inclusion of select antimicrobials such as ionophores (FDA approval pending for lactating dairy cows) along with supplemental unsaturated fats may decrease the cleavage of glycerol from the triacylglycerol molecule by ruminal bacteria, thus, allowing greater delivery of linoleic acid to the small intestine for absorption (102). Reacting butylamine with soybean oil to produce butylsoyamide resulted in a ruminally inert mixture of fatty acids, which increased linoleic acid concentration in plasma and milk compared with that of untreated soybean oil when fed to lactating dairy cows (53). Treatment of whole canola seeds with alkaline H_2O_2 resulted in a greater amount of unsaturated fatty acids flowing to the duodenum of steers than feedng untreated whole canola seeds (51). Protection of dehulled cottonseeds with protein-aldehyde complexes (Protected Lipid; Rumentek Industries, Australia) delivered approxi-

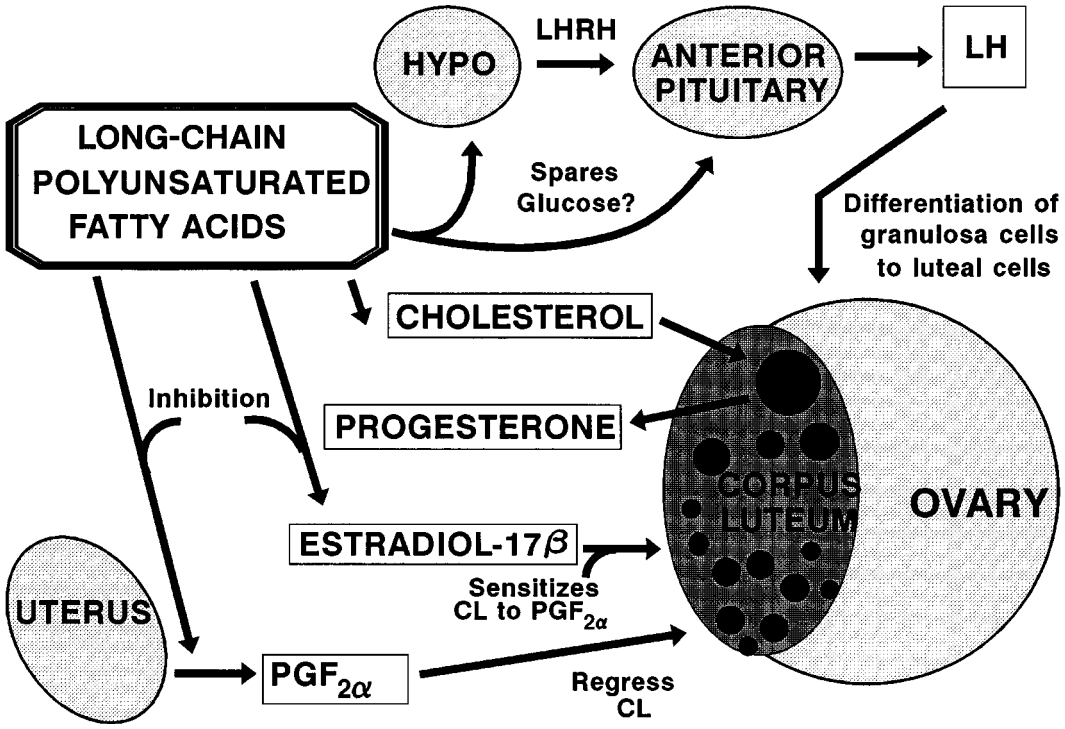

Figure 2. Suggested mechanisms of action by which supplemental long-chain polyunsaturated fatty acids (PUFA) improve conception rates of lactating dairy cows include the following: 1) PUFA may spare glucose to stimulate release of LH from the anterior pituitary gland, which stimulates development of ovarian luteal cells, 2) PUFA may increase circulating concentrations of cholesterol, a precursor of progesterone that is associated with improved fertility, and 3) PUFA may inhibit production of $PGF_{2\alpha}$ and estradiol-17β in order to increase the lifespan of the corpus luteum (CL), potentially improving survival of the embryo. LHRH = LH-releasing hormone.

mately 175 g/d of linoleic acid to the lower gut of lactating Hereford cows. Overall pregnancy rates were improved from 63 to 79% (105). The advantages of diets that are high in linoleic acid on reproductive performance may be greater during periods of heat stress because $PGF_{2\alpha}$ synthesis is increased during that time (107).

Growing evidence indicates that the design and delivery of supplemental fatty acids to the lower gut may target reproductive tissues to improve reproductive function and fertility. Changes in follicular dynamics and CL function, as documented by fat supplementation, may be due to alterations in metabolic hormones like IGF-I and growth hormone or hormonal clearance. Improvement in embryo survival may be associated with the suppression of uterine prostaglandin secretion via linoleic acid or other longer chain unsaturated fatty acids.

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